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Chronic CAD/Stable Ischemic Heart Disease

SNP STATUS AT 9P21.3 DOES NOT PREDICT POST-OPERATIVE MORTALITY IN PATIENTS UNDERGOING CABG

ACC Moderated Poster Contributions
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Session Title: New Twists with Older Risk Markers in SIHD
Abstract Category: 2. Chronic CAD/Stable Ischemic Heart Disease: Clinical
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Background: The contribution of genetics to coronary heart disease (CHD) is incompletely understood, including the precise role of the risk-associated single nucleotide polymorphisms (SNP) at chromosome 9p21.3. In most populations, 9p21.3 predicts CHD onset but not subsequent outcome. In contrast, a recent publication reported that 9p21.3 variant carriage predicts increased mortality risk after bypass surgery (CABG), but those results have not been replicated. Here, we sought to examine the association of 9p21.3 SNPs with mortality among patients who underwent CABG at Intermountain Healthcare.

Methods: Consenting patients (average age 58.1, 78% male) were genotyped for rs2383206 and followed post-CABG for a mean of 5.3 +/- 3.1 years to compare genetics of those who died (n=90) with those who survived (N=362). As per the recent report, the recessive model was evaluated using Kaplan-Meier methods, and the Cox proportional hazards regression was used to estimate hazard ratios (HR) and 95% confidence intervals (CI) with adjustment for demographics, clinical, and treatment variables. Additional testing was performed for the composite outcome of MI or death, and secondary analyses used the additive genetic model.

Results: In recessive modeling, no difference in mortality was found between those with 2 copies of rs2383206 and those with 0-1 copy (log rank p=0.98; adjusted HR=0.95, CI=0.60, 1.5, p=0.82). Using the more traditional additive genetic modeling, differences also were not significant in univariable (log rank p=0.59) and multivariable analysis (HR=0.94, p=0.69). Neither the recessive nor the additive model was statistically significant for the composite outcome of death and MI (p=0.48, p=0.29, respectively).

Conclusion: The 9p21.3 SNP rs2383206 did not predict death or the composite of MI and death across multiple inheritance models in our population of patients undergoing CABG. The utility of this marker therefore appears to be limited to predicting the onset/presence of coronary atherosclerosis, as in earlier reports, but not subsequent mortality risk. The genetic contribution to CHD and its implications are complex, remain incompletely understood, and warrant further study.